

U.S. Department of Labor
Office of Administrative Law Judges
800 K Street, N.W.
Washington, D.C. 2001-8002

In the Matter Of

HATTIE WOODS

Claimant

V.

CONSOLIDATION COAL COMPANY

Employer

and

DIRECTOR, OFFICE OF WORKERS'

COMPENSATION PROGRAMS.

Party In Interest

Appearances:

Frederick K. Muth, Esquire

Hensley, Muth, Garton & Hayes

For the Claimant

Mary Rich Malloy, Esquire

Jackson & Kelley, PLLC

For the Employer

Before: STUART A. LEVIN

Administrative Law Judge

DECISION AND ORDER

This proceeding arises from a claim for survivor benefits filed pursuant to the Black Lung Benefits Act, as amended, 30 U.S.C. § 901, *et. seq.*, (the “Act”), and the regulations promulgated thereunder, 20

C.F.R. Parts 718 and 725.

Benefits are awarded under the Act to eligible survivors of coal miners whose deaths were due to or substantially contributed to by pneumoconiosis, a dust disease of the lungs arising from coal mine employment, and commonly known as black lung disease. 20 C.F.R. §718.201.

Following notice to all interested parties, and in accordance with the provisions of 20 C.F.R. Part 725 and 29 C.F.R. Part 18, a formal hearing was held before me in this matter on September 27, 2000 at Pipestem, West Virginia. Each party was afforded the opportunity at such time to present their evidence and arguments. The record was left open for thirty (30) days to permit the taking of the deposition of Dr. Donald L. Rasmussen and the filing of briefs. These matter have now been submitted. Without objection, the deposition is admitted into evidence as Claimant's Exhibit (CX) 3.

As discussed at the hearing, there is some confusion in this case regarding the numbering of the Director's exhibits. My review of the file transmitted by the District Director discloses that there were 29 exhibits developed in regard to the survivor's claim through the March 10, 2000 letter to the Claimant advising her of an award of interim benefits. There was then added to the file the exhibits developed in connection with the three claims which were filed by the miner. Irrespective of how they have been marked by the District Director's staff, I will consider Director's Exhibit (DX) 30, 31 and 32 to be the documents developed in connection with the 1980, 1985 and 1993 claims, respectively. I will consider the documents associated with the transmittal of the case to be DX 33.

The record in this then case consists of DX 1-33; CX 1-3; Employer's exhibits 1-10 ("EX 1-10") and the testimony of the Claimant.

PROCEDURAL HISTORY

I have noted that the miner filed three claims during his lifetime. The first two were denied. The third claim was allowed by Administrative Law Judge Samuel J. Smith. In finding that the miner had established the existence of pneumoconiosis pursuant to §718.202(a)(4) Judge Smith held:

"Contrary to the opinions of the physicians who found CWP to be absent, the miner's obstructive respiratory impairment, his emphysema and bronchitis, can constitute CWP if it is substantially aggravated by, or significantly related to, his coal mine employment. In this respect Dr. Rasmussen gave convincing testimony with regard to the etiology of the Claimant's respiratory impairment. He found the miner to be suffering from coal workers' pneumoconiosis, as well as emphysema, citing the miner's two obvious risk factors, i.e., his tobacco abuse and his work history, for the miner's respiratory insufficiency."

Citing *Warth v. Southern Ohio Coal Co.* 60 F3d. 173 (4th Cir. 1995) (holding that obstructive lung

disease is encompassed within the definition of pneumoconiosis) the Benefit Review Board upheld Judge Smith's finding under §718.204 (a)(4) both in its initial Decision and Order in the matter as well as in its denial of the Employer's motion for reconsideration.

The miner died on January 9, 1999 and his surviving spouse filed the instant claim on February 15, 1999. This claim was ultimately approved by the District Director following an informal conference. The Employer has controverted the claim and the matter is now before me for a *de novo* consideration.

THE ISSUES

The Employer contends, *inter alia*, that the biopsy and CT scan evidence developed subsequent to the miner's award of benefits shows that he did not have pneumoconiosis. Claimant maintains that the Employer is collaterally estopped from raising the issue of whether the miner was suffering from pneumoconiosis at the time of his death.

As Claimant's counsel notes the United States Court of Appeals for the Fourth Circuit held in *Sedlack v. Braswell Services Group*, 134 F. 3d 219 (4th Cir. 1998):

"Collateral estoppel" forecloses 'the relitigation of issues of fact or law that are identical to issues which have been actually determined and necessarily decided in prior litigation in which the party against whom [issue preclusion] is asserted had a full and fair opportunity to litigate' *Ramsay v. INS* 14 F. 3d 206 (4th Cir. 1994) (quotation omitted). For collateral estoppel to apply, the proponent must establish that: (1) the issue sought to be precluded is identical to the one previously litigated; (2) the issue must have been actually determined in the prior proceeding; (3) determination of the issue must have been a critical and necessary part of the prior proceeding; (4) the prior judgement must be final and valid; and (5) the party against whom estoppel is asserted must have had a full and fair opportunity to litigate the issue in the previous forum."

The Board has indicated that the above elements are applicable in claims for survivor benefits where the miner had been awarded disability benefits and, accordingly, the existence of pneumoconiosis was a critical and necessary part of his entitlement to such award. *Hughes v. Clinchfield Coal Co.*, 21 B.L.R. 1-134 (1999). However, the Board also held that the party against whom estoppel is being asserted may not have had a full and fair opportunity to litigate the issue of the existence of pneumoconiosis where autopsy evidence was not available and could not have been adduced at the time the miner's claim was adjudicated.¹

¹I also note and am persuaded by the Board's unpublished decision in the case of *Ward v. Circle A&G* (99-0344 BLA) (1999) (<http://www.dol.gov/dol/brb/public/cases/blklung/unpublished/Dec99/99-0344.htm>), wherein the Board specifically found that the doctrine of

There was no autopsy in the instant case. There was a biopsy conducted subsequent to the miner's award of benefits which reportedly failed to show the existence of pneumoconiosis. But, as Claimant's counsel points out, pursuant to §718.106 (c), negative biopsy evidence is not to be considered conclusive evidence that the miner did not have pneumoconiosis. Indeed, Employer's counsel acknowledges that "the slides may not conclusively rule out the presence of pneumoconiosis (the samples are small)..." The other evidence on which the Employer relies includes readings of CT scans and x-rays of the miner's chest taken subsequent to the award of disability benefits. The Board has not carved out an exception to the collateral estoppel rule based on such evidence. Furthermore, it was Judge Smith's finding that the existence of "legal" pneumoconiosis had been established in the miner's claim that led to the Board's upholding of the award.

The Employer had a full and fair opportunity to litigate the issue of the existence of pneumoconiosis under §718.202 (a)(4) during its contest of the miner's claim. The finding of the existence of pneumoconiosis was critical and necessary to the award of disability benefits. The award had become final following the Employer's failure to timely seek judicial review of the Board's decision. Accordingly, I conclude that collateral estoppel is applicable to the issue of the existence of pneumoconiosis and decline to reconsider such issue in this case.

The sole issue to be decided, then, is whether the miner's death was caused or contributed to by his pneumoconiosis.

STATEMENT OF THE CASE

The evidence in this case includes records from the Princeton Community Hospital, Dr. G. Duremdes, Dr. R. Chambers, Dr. John Muldoon, Dr. E. R. Jabour and Dr. C. E. Gabe. These are to the combined effect that in or about the summer of 1997, the miner developed pain in his upper right chest wall and shoulder. A CT scan suggested a mass in the right upper quadrant of the lung. A needle biopsy of the area was non diagnostic as was a bronchoscopy and transbronchial biopsy. It was then decided to do an open biopsy and/or surgical excision.

Prior to the surgery the miner was evaluated by Dr. Jabour on January 5, 1998. This included a review of pulmonary function studies which had been conducted on December 31, 1997. Dr. Jabour noted that these disclosed a vital capacity of 73% of predicted, a FEV1 of 46% of predicted and a DLCL of 27%. It was noted on the report of this study that the miner had ceased 40 years, one pack per day of cigarette smoking two weeks previously. Dr. Jabour opined that the FEV1 of 1.38 left enough margin that a right upper lobe resection could be performed but the severely reduced DLCL increased the miner's risk for surgery as a tissue or lung sparing procedure. He recommended that a wedge resection

collateral estoppel was applicable in a survivor's claim when the miner had been awarded benefits and there was no autopsy evidence submitted.

should be performed if at all possible.

The surgery was conducted on January 13, 1998 by Dr. Gene Duremdes with the assistance of Dr. Generoso Duremdes. The miner was found to have a tumor mass occupying the whole right upper lobe with extensive adhesions to the right upper chest wall. Findings indicative of long standing smoking were noted in the middle and lower lobes. Multiple biopsies revealed squamous cell carcinoma. It was observed by the surgeons that since the tumor was extensive and unresectable and since the patient cannot tolerate a pneumonectomy and since the tumor had infiltrated the right apex toward the large vessels and axillary plexus, no further surgery was planned.

In a consultation report, dated March 24, 1998, by Dr. Charles E. Gabe, a Radiation Oncologist, it was noted that the miner had embarked on a period of chemotherapy which was discontinued because of severe, consistent nausea which was refractory to antiemetics. He then had radiation therapy under the care of Dr. Gabe. It was noted on May 21, 1998 that the miner had tolerated this treatment well and had a nice response relative to cytoreductive effect. However, on August 12, 1998, Dr. Gabe reported that the miner continued to have a progressive large, invasive malignancy, he had not responded to the radiotherapy and his situation is terminal.

The miner was rehospitalized at Princeton Community in January 1998 for marked weakness due to cachexia of malignancy and severe chronic obstructive lung disease (COPD), in July 1998 for an exacerbation of COPD, and in September 1998 for management of pain.

Dr. Muldoon's office notes indicate that he began treating the miner in June 1996 and continued to do so, both on an inpatient and outpatient basis, until October 1998. His diagnosis during these visits included COPD, which at times was described as severe.

The final hospitalization occurred on December 6, 1998 because of a syncopal episode which was thought to be due to anemia. The secondary diagnoses at this time included pneumonia requiring antibiotics, progressing squamous cell carcinoma of the right upper lobe, head contusion and coal workers' pneumoconiosis. The miner decided to leave the hospital against medical advice on the day after admission. It was noted that Dr. Long was the miner's medical doctor and had agreed to do home visits.

The record includes a Physician Order Form from Appalachian OH-9, Inc. which notes that the miner was transferred to Dr. Long's services beginning December 22, 1998. What appears to be Dr. Long's treatment records show that he saw the miner at home on January 3, 1999 when he recorded his blood pressure and prescribed medication..

The miner's death certificate indicates that he died at home on January 9, 1999. The immediate cause of death is listed as cancer of the lung due to respiratory arrest due to occupational pneumoconiosis. "Chronic Cancer of the Lung" was also listed as an "other significant condition

contributing to death but not related to the underlying cause.” The certificate was certified by Dr. Marshall C. Long, whose signature in item 23 indicated that he was not present at the time of death. There was no autopsy.

In a statement dated June 7, 1999, Dr. Long reported:

“George W. Woods suffered from coal workers’ pneumoconiosis for many years and had multiple X rays done. However no autopsy was done post mortem. If you have any questions feel free to contact my office...”

On June 11, 1999, a DOL claims examiner wrote to Dr. Long, enclosed copies of pertinent medical records and requested his opinion as to whether black lung disease hastened the miner’s death and if so, how did this occur? He was asked also to explain why the death certificate listed chronic cancer of the lung as an other significant condition. There is no record of a response from Dr. Long to this inquiry.

Dr Jabour issued the following statement on September 14, 1999:

“Mr. Wood’s death was en hastened by the presence of pneumoconiosis. Chest x-ray dated 5-4-94 revealed pneumoconiosis, Category P/Q2/2 with bilateral chest wall thickening. PFT dated 12-31-97 revealed a FEV1 of 46%; DLCO of 27%; TLC of 98% and FVC Of 73%. Patient suffered from pneumoconiosis and this did contribute to his death.”

The Employer has submitted the reports, supplemental reports and/or depositions of Dr. Jerome Wiot, Dr. Echols A. Hansburger, Dr. Stephen T. Bush, Dr. Richard Naeye, Dr. George Zaldivar, Dr. Peter Tuteur, Dr. George Fino and Dr. James Castle. Dr. Wiot is board certified in radiology, Drs. Hansbarger, Bush and Naeye are board certified in pathology and Drs. Zaldivar, Tuteur, Fino and Castle are board certified in pulmonary diseases. With the exception of Dr. Wiot, each of these physicians have reviewed extensive medical data of record pertaining to the miner dating from the development of his claim for benefits to his death.

Dr. Wiot’s report relates to his interpretations of CT scans of November 7, 1997 and August 6, 1998 in regard to whether “medical” pneumoconiosis is shown.² For reasons already stated, his opinion is not relevant to the present inquiry.

For the same reasons, Drs. Hansburger, Bush and Naeye reports, to the extent they relate to the absence of “medical pneumoconiosis” in biopsy material, are not material to the issue involved in this

²I note that Dr. Wiot did find evidence of emphysema on the scans.

case.

After noting that there is no evidence of occupational pneumoconiosis of any variety, Dr. Hansbarger opined in his December 29, 1999 report that it “appears from the medical record that the miner died as a result of epidermoid carcinoma of the lung.” He further opined that his death was not contributed to by coal workers’ pneumoconiosis or hastened in any way by his history of coal mine employment “because it appears from the medical record that Mr. Woods did not suffer from coal workers’ pneumoconiosis.” He attributed the miner’s chronic obstructive pulmonary disease to his long history of cigarette smoking. He did not offer an opinion as to what role, if any, the COPD played in the miner’s death.

After finding that the biopsy tissue failed to show the presence of pneumoconiosis, and opining that if it were present in other areas of the lung, it would be very mild, Dr. Bush concluded that pneumoconiosis or occupational exposure to coal dust did not contribute to respiratory impairment or disability. He noted that chronic obstructive lung disease (emphysema) may have caused respiratory impairment to some degree during the miner’s lifetime but offered no opinion as to whether the condition caused, contributed to or hastened the miner’s death.

Dr. Naeye noted the results of the December 1997 pulmonary function studies and stated that, if valid, they suggest some degree of airway obstruction with no evidence of restrictive fibrosis. He then went on to state that there was no objective evidence of “CWP” and, being absent, CWP could not have caused any abnormalities of lung function, disability or hastened death.

Dr. Tuteur found the available data to be “most significant in the diagnosis of a right upper lobe mass as a manifestation of carcinoma of the lung which at the time of the diagnosis, was unresectable due to local chest wall metastases as well as malignant pleural effusion. “ He noted further that subsequent radiotherapy and chemotherapy was unsuccessful and after a “one year generally downhill course, he died with and because of carcinoma of the lung, its metastases, its sequelae, and its treatment.” After opining that there is insufficient evidence to justify a diagnosis of pneumoconiosis, Dr. Tuteur went on to state that the miner did have primary pulmonary problems, including chronic obstructive lung disease with bullous emphysema and that the impairment was progressively worsening. Yet, he was of the opinion that inhalation of coal mine dust played no role in the development of the impairment or in the miner’s death.

Dr. Zaldivar issued a report on May 23, 2000 in which he offered the following opinions:

1. There is no evidence in any of these records to justify a diagnosis of coal workers pneumoconiosis.
2. There was a respiratory impairment present prior to his death. The respiratory impairment was due to bullous emphysema caused by smoking. None of the

pulmonary impairment was due any pneumoconiosis since no pneumoconiosis was present.

3. From the pulmonary standpoint, prior to his death Mr. Woods was unable to perform any work.

4. Coal dust exposure did not play any role in the pulmonary disability prior to his death. The pulmonary disability was due to bullous emphysema caused by smoking.

5. Neither coal worker's pneumoconiosis nor coal dust exposure played any role in his death, nor did it hasten his death. Mr. Woods did not have coal worker's pneumoconiosis nor any dust disease of the lungs.

6. Even if Mr. Woods were found to have coal workers' pneumoconiosis, which in my opinion he does not have, the cause of his death was lung cancer. The lung cancer was a squamous carcinoma which is not a manifestation of coal dust disease of the lungs nor coal worker's pneumoconiosis. Cancer of the lungs has never been linked to coal mine work. Therefore whether or not Mr. Wood had coal workers' pneumoconiosis, death was caused by the cancer which was causing his malnutrition and was responsible for the need of large amount of medications which was blunting the respiratory centers in the brain. Originally, there are some records suggesting supraventricular tachycardia with fainting spells. This meant that the heart probably was involved with cancer as well, or else he had developed coronary artery disease. Neither one of these problems were related to any occupation. Therefore, it is my opinion that Mr. Woods would have died when and as he did even if he had never worked in a coal mine.

The major portion of Dr. Fino's August 14, 2000 report is devoted to a documented discussion to the effect that coal mine dust exposure does not cause obstructive lung disease. He did opine that the miner clearly died from cigarette smoking as this is what caused his cancer.

Dr. Castle opined in his August 21, 2000 report that there was no physical, physiologic, radiographic or pathologic evidence that the miner had coal workers' pneumoconiosis. He attributed the miner's total disability prior to his death to tobacco induced pulmonary emphysema. He concluded that the miner died as a result of extensive squamous cell carcinoma of the lung due to cigarette smoking and that he would have died exactly when he did had he never stepped foot inside a coal mine and regardless of his occupational history.

Drs. Fino, Zaldivar and Tuteur issued supplemental reports in apparent response to an inquiry from Employer's counsel in which they were asked whether, assuming that the miner had "legal pneumoconiosis (a chronic dust disease of the lungs resulting in pulmonary impairment significantly related to or aggravated by coal dust exposure)," their opinions would change as to whether

pneumoconiosis caused, contributed to or hastened the miner's death. Drs. Zaldivar and Fino responded simply that their opinions would not change. Dr Tuteur responded, in significant part:

"In sum, my opinion would not change. Specifically, it must be recognized that Mr. George Woods died with and because of carcinoma of the lung that was widely metastatic. At the time of diagnosis, it was unresectable because of local chest wall metastases and malignant pleural effusion. He was considered in sufficient and adequate health to undergo not only radiotherapy, but also multi-drug chemotherapy. Because of the biological nature of the tumor, these therapies were unsuccessful. His clinical course was downhill gradually over the next year. His death was accounted for not only by the carcinoma of the lung, its metastases, its sequelae, but also treatment."

...

Dr. Tuteur went on to note that the miner's lowest measured FEV-1 at the time of the diagnosis of malignancy was above 1.25 liters, and that it is well recognized that longevity is unaffected and unchanged until the FEV-1 measurement drops below this level.

The Claimant has submitted a report from Dr. Rasmussen, dated June 9, 2000, in which he noted having reviewed the evidence of record and opined, in pertinent part:

"In the case of Mr. Woods, he did have progressive deterioration in his respiratory function and was in chronic respiratory failure for some time prior to his demise. He had multiple hospitalizations for pneumonia, exacerbation of COPD, and was on chronic oxygen therapy at 3 liters a minute.

"There is no evidence that coal mine dust exposure is associated with increased risk of carcinoma of the lung.

"Mr. Woods' carcinoma of the lung failed to improve with chemotherapy and radiation therapy. Both his chemotherapy and his radiation therapy could have further injured his lungs. Those factors combined with the cancer itself could clearly have worsened Mr. Wood's lung function, however, had Mr. Woods had normal lungs, the insult to his lung tissues would not have produced the fatal respiratory failure which did cause his demise.

"Based on all of the above, it remains my opinion that Mr. Woods did suffer from coalworkers' pneumoconiosis which resulted in a severe and totally disabling respiratory insufficiency and which clearly hastened his death by virtue of rendering him incapable of sustaining the impact of his carcinoma and therapy."

At his deposition, taken on September 18, 2000, Dr Zaldivar testified, in substance, that his cancer had caused the miner to become malnourished, had “sapped all his strength,” and simply “broke down his entire body.” It was this fact, together with the palliative medication he was receiving, which led to the miner’s death. He maintained that the miner’s other health problems, including his emphysema, neither affected his ability or inability to fight cancer, nor otherwise play any special role in the miner’s death. Dr. Zaldivar noted in this regard that by the time the cancer was found it was too far advanced to be operable irrespective of the fact that his lungs were “very bad.” He opined that there was no evidence in the record that there was a fatal respiratory failure which caused death. Rather, the miner died because his whole body, including the lungs, heart and brain, failed.

Dr. Castle was deposed on September 5, 2000. He testified, in substance, that the miner died in January 1999 from cancer which was shown on x-ray in 1997. He noted that most people with untreated lung cancer will live anywhere between six to 30 months and that the miner's clinical course was exactly what would be expected of an individual with this type of lung cancer. He expressed disagreement with Dr. Rasmussen's opinion that the miner's severe and disabling respiratory impairment rendered him incapable of sustaining the impact of his carcinoma and its therapy. He testified:

The thing that caused his demise was the fact that his tumor was one that did not respond well to therapy, was not in a location where it could be treated adequately but progressed as all tumors of this type do regardless of any underlying lung disease.

"He would have died as and when he did even if he only had one lung."

Dr. Naeye's deposition was, for the most part, confined to the issue as to whether or not the miner had pneumoconiosis.

When deposed on October 12, 2000, Dr. Rasmussen was asked: "when a person has severe respiratory disease, that is chronic respiratory disease, and they contract something like cancer, how does the underlying respiratory disease affect the physician's ability to treat his patient," to which responded as follows:

Well, in some cancers it wouldn't necessarily make much difference but when you have a pulmonary cancer, first of all, severe lung disease would prevent any potential curative surgical procedures, that is one clear example, in addition, this was not operable, this case, such treatments as radiation therapy and chemotherapy could actually further damage even a healthy lung and could certainly clearly equally damage an already diseased lung, so that makes treatment — even nonsurgical treatment is at a greater risk for further damage to the lung."

When asked whether there is an indication that a more vigorous type of therapy was withheld because of the above consideration, Dr. Rasmussen responded:

"I think that as I read the records the patient's family discussed with the physicians the idea of using chemotherapy and the chemotherapy was not continued. He did receive radiation therapy and Methotrexate which is a form of cancer therapy."

FINDINGS AND CONCLUSIONS

This claim was filed after January 1, 1982. Consequently, pursuant to §718.205 (c) of the regulations, to be entitled to benefits the miner's surviving spouse must establish that any of the following criteria is

met:

- (1) Competent medical evidence establishes that pneumoconiosis was the cause of the miner's death or
- (2) Pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis, or
- (3) The miner is shown to have complicated pneumoconiosis

Survivors are not eligible for benefits, however, where the miner's death was caused by a traumatic injury or the principle cause of death was a medical condition not related to pneumoconiosis, unless evidence establishes that pneumoconiosis was a substantially contributing cause of death.

The United States Court of Appeals for the Fourth Circuit has held that any condition which hastens a miner's death is a substantially contributing cause of death for the purpose of §718.205. *Shuff v. Cedar Coal Company*, 967 F.2d 977 (4th Cir. 1992).³ The proviso has now been incorporated in §718.205 (c)(5) by the amendments to the regulations which became effective on January 19, 2001.

Pneumoconiosis had been defined at §718.201 as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. The definition includes such diseases as coal workers' pneumoconiosis and silicosis and any other chronic pulmonary disease significantly related to or substantially aggravated by dust exposure in coal mine employment.

Thus, as noted by the Fourth Circuit in *Barber v. Director, OWCP*, 43 F.3d (4th Cir., 1995):

“We have reminded ALJs and the BRB on several occasions that ‘pneumoconiosis’ is a legal term defined by the Act, and they must bear in mind when considering medical evidence that physicians generally use ‘pneumoconiosis’ as a medical term that comprises merely a small subset of afflictions compensable under the Act. If there is any lingering confusion on this point, let us dispel it now. The legal definition of ‘pneumoconiosis’ is incorporated into every instance the word is used in the statute and regulations.”

(Footnotes omitted. Emphasis supplied)

This principle has also been incorporated in the regulatory amendments at §718.201 which now defines pneumoconiosis as follows:

- (a) For the purpose of the Act, “pneumoconiosis” means a chronic dust disease of the

³As the miner was reportedly last employed at a coal mine in West Virginia, any judicial review of this claim would come under the jurisdiction of the Fourth Circuit.

lung and its sequale, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or “clinical”, pneumoconiosis and statutory, or “legal” pneumoconiosis.

(1) Clinical Pneumoconiosis. “Clinical pneumoconiosis” consists of those diseases recognized by the medical community as pneumoconiosis, i.e, the condition characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers’ pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.

(2) Legal Pneumoconiosis. “Legal pneumoconiosis” includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

(b) For the purpose of this section, a disease “arising out of coal mine employment” includes any chronic pulmonary disease or respiratory impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

(c) For the purpose of this definition, “pneumoconiosis” is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

Another pertinent amendment to the regulations involves the weight to be afforded a “treating physician’s” opinion as set forth in §718.104 (d) as follows:

(D)Treating physician. In weighing the medical evidence of record relevant to whether the miner suffers, or suffered from pneumoconiosis, whether the pneumoconiosis arose out of coal mine employment, and whether the miner is, or was, totally disabled by pneumoconiosis or died due to pneumoconiosis, the adjudication officer must give consideration to the relationship of any treating physician whose report is admitted into the record. Specifically, the adjudication officer shall take into consideration the following factors in weighing the opinion of the miner’s treating physician:

(1) Nature of relationship. The opinion of a physician who has treated the miner for respiratory or pulmonary conditions is entitled to more weight than a physician who has treated a miner for non-respiratory conditions.

(2) Duration of relationship. The length of the treatment relationship demonstrates whether the physician has observed the miner long enough to obtain a superior understanding of his or her condition.

(3) Frequency of treatment. The frequency of physician-patient visits demonstrates whether the physician has observed the miner often enough to obtain a superior understanding of his or her condition; and

(4) Extent of treatment. The types of testing and examinations conducted during the

treatment relationship demonstrates whether the physician has obtained superior and relevant information concerning the miner's condition;

(5) In the absence of contrary probative evidence, the adjudication officer shall accept the statement of a physician with regard to the factors listed in paragraphs (d)(1) through (4) of this section. In appropriate cases, the relationship between the miner and his treating physician may constitute substantial evidence in support of the adjudication officer's decision to give that physician's opinion controlling weight, provided that the weight given to the opinion of a miner's treating physician shall also be based on the credibility of the physician's opinion in light of its reasoning and documentation, other relevant evidence and the record as a whole.

These clarifications of existing law demonstrate on the record that the miner's respiratory impairments, including obstructive respiratory disease and emphysema, were caused, in part, by coal mine dust exposure and thus constituted "legal" pneumoconiosis. It follows that to the extent that they have relied on their conclusions that the miner did not have "legal" or "medical" pneumoconiosis, the opinions of the Employer's medical experts must be rejected.

Both the miner's treatment records and the consensus of the medical experts establish that the miner's pneumoconiosis was severely disabling during the last year of his life. Nevertheless, this fact, standing alone, is no longer a sufficient basis for awarding survivor benefits. The miner's pneumoconiosis must have caused, contributed to or hastened the miner's death.

I first turn to the death certificate. The certificate indicates that Dr. Long was not in attendance when the miner died and there was no autopsy. The listing of the causes of death is confusing and would appear to point to pneumoconiosis as a cause of the cancer. However, Dr. Rasmussen and the Employer's medical witnesses are in agreement that pneumoconiosis does not cause lung cancer. Although requested to do so, Dr. Long has declined to clarify or support his certification of the cause of death.

Dr. Jarboe had treated the miner at one time. However, I find no indication that he had seen him during the last year of his life. In any event, his opinion that pneumoconiosis contributed to and hastened the miner's death appears to be based solely on the fact that the miner had x-ray evidence of pneumoconiosis and impaired pulmonary functioning. He offers no explanation as to how these factors contributed to or hastened the miner's death. Accordingly, I reject his opinion also.

While Dr. Rasmussen offered a detailed explanation as to why he believed the miner had "legal pneumoconiosis," I find his reasoning as to how this contributed to his death to be lacking in clarity and not supported by the evidence. As I interpret Dr. Rasmussen's report and deposition, he appears to be suggesting that the miner's pneumoconiosis interfered with the treatment he received for his cancer or made it less effective. Yet the record shows that surgical excision of the tumor was contraindicated because it had already involved the chest wall, chemotherapy was halted because of the intractable

nausea, and radiation treatment was stopped because it simply was not effective. Nowhere in this record do I find that any of these treatments were discontinued because the miner could not tolerate them due to the pulmonary impairment stemming from his pneumoconiosis. In fact, Dr. Gabe observed that the miner well tolerated his treatment.

I note also that Dr. Rasmussen appears to accept the miner's dying in respiratory arrest as a given in this case, apparently based on the death certificate. I find Dr. Zaldivar's explanation as to how death occurred to be more convincing, i.e., that the miner died from a total body collapse and that this, in turn, was due to the debilitating effects of the cancer. Considering the record as a whole, I am convinced that the miner's death would have occurred when and how it did if he never had exposure to coal mine dust.

The Claimant has the burden of proving her entitlement by the preponderance of the evidence. As the evidence does not establish that the miner's death was caused, contributed to or hastened by pneumoconiosis, she has failed to carry this burden. Consequently, her claim must be denied.

ORDER

The claim of Hattie Woods for survivor benefits under the Black Lung Benefits Act is hereby denied.

STUART A. LEVIN
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of service of this Decision by filing Notice of Appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C., 20013-7601 (20 CFR 725.481). A copy of the Notice of Appeal must also be served on Donald Shire, Esquire, Associate Solicitor, Room N-2605, 200 Constitution Avenue, N.W., Washington, D.C. 20210